Research Article

Early-Onset Mirizzi Syndrome Case With Cholecystocholedochal Fistula in A Young Female: Multimodality Imaging and Interventional Radiology As A Bridge To Definitive Surgery

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Abstract

Background: Mirizzi syndrome is an uncommon complication of long-standing cholelithiasis in which a stone impacted in the gallbladder neck or cystic duct obstructs the common hepatic duct. It is reported in approximately 0.05% to 4% of patients treated for gallstone disease, occurs predominantly in women, and is usually encountered from the fifth decade of life onwards. Presentation in a patient under 40 years of age that has already progressed to cholecystocholedochal fistula is seldom documented. **Case presentation:** A 33-year-old woman presented with six months of right upper quadrant pain that had intensified over the preceding week, accompanied by progressive jaundice, malaise, anorexia, vomiting, acholic stools and dark urine, together with 4 kg of weight loss. Laboratory testing showed total bilirubin 16.4 mg/dL with direct bilirubin 13.5 mg/dL, aspartate aminotransferase 171 U/L, alanine aminotransferase 106 U/L, leukocytes 12,700/mm3, C-reactive protein 2.86 mg/dL, haemoglobin 8.3 g/dL, hypoalbuminaemia and CA 19-9 of 234 U/mL with a normal carcinoembryonic antigen. Ultrasonography demonstrated an echogenic focus of about 1.7 cm at the level of the common hepatic duct with bilateral intrahepatic duct dilatation, and computed tomography showed hepatomegaly with small hepatic abscesses, a thick-walled gallbladder containing sludge and common bile duct wall thickening. The findings were interpreted as Mirizzi syndrome complicated by acute cholangitis. **Management and outcome:** An interventional radiology-first strategy was adopted. Percutaneous transhepatic biliary drainage was performed through a right intrahepatic duct. Cholangiography confirmed extrinsic compression of the common hepatic duct by a stone at the gallbladder neck, with a filling defect attributed to intraductal clot. Total bilirubin fell from 16.4 to 6.2 mg/dL within several days and the inflammatory markers improved. Open cholecystectomy with common bile duct exploration was subsequently undertaken and disclosed a cholecystocholedochal fistula corresponding to Csendes type II to III. The stone was extracted, the defect was repaired with a gallbladder remnant flap and a T-tube was placed. Histopathology showed chronic cholecystitis with acute exacerbation and Rokitansky-Aschoff sinuses, without malignancy. Total bilirubin declined further to 3.3 mg/dL and the patient was discharged without major complications. **Conclusion:** Mirizzi syndrome should remain in the differential diagnosis of obstructive jaundice in young adults, particularly when marked cholestasis coexists with a raised CA 19-9 and no mass lesion on imaging. Staged management, in which percutaneous biliary decompression precedes definitive hepatobiliary surgery, allowed sepsis control and metabolic stabilisation before an operation on a distorted biliary hilum.

Keywords: Mirizzi Syndrome, Cholecystocholedochal Fistula, Percutaneous Transhepatic Biliary Drainage, Obstructive Jaundice, CA 19-9, Interventional Radiology.

1 Introduction

Mirizzi syndrome describes obstruction of the common hepatic duct by a gallstone impacted in Hartmann pouch or the cystic duct, producing obstructive jaundice and progressive biliary inflammation [1]. Although cholelithiasis is common worldwide, Mirizzi syndrome accounts for only about 0.05% to 4% of patients with symptomatic gallstone disease [1], [2]. Its frequency varies geographically: the syndrome is identified in roughly 0.5% of cholecystectomies performed in Europe, whereas series from Asia and from Central and South America report rates approaching 4.7% to 5.7% [1]. Women account for 55.6% to 77% of reported patients, a distribution that reflects the higher prevalence of gallstone disease in women [1], [2]. Advanced age, recurrent cholecystitis and delayed operative treatment of symptomatic cholelithiasis are the risk factors most consistently emphasised, and the condition is therefore encountered predominantly in middle-aged and older patients [2].

Preoperative recognition remains difficult despite advances in hepatobiliary imaging, because the clinical and radiological picture overlaps with choledocholithiasis, benign biliary stricture and cholangiocarcinoma [1], [2]. Patients typically report right upper quadrant pain and jaundice, with nausea, vomiting, fever and features of cholangitis in a substantial minority [1], [3]. Laboratory testing usually shows a cholestatic pattern with hyperbilirubinaemia, raised alkaline phosphatase and gamma-glutamyl transferase, and a variable inflammatory

response [1]. CA 19-9 may also rise steeply in the presence of severe cholestasis or active biliary infection, which can generate unwarranted suspicion of malignancy [4], [5].

Imaging determines both the diagnosis and the operative strategy. Abdominal ultrasonography detects gallstones, gallbladder wall thickening and intrahepatic duct dilatation, but it visualises extrinsic ductal compression poorly and its reported sensitivity for Mirizzi syndrome ranges from around 10% to 50% [1], [6]. Cross-sectional techniques delineate biliary anatomy more reliably, and magnetic resonance cholangiopancreatography reaches a sensitivity of approximately 80% with a specificity of 83% in comparative work [1], [7]. Even so, a large proportion of cases are still identified only at operation, with preoperative detection rates across published series varying between 12% and 84% [1], [8]. Late recognition is clinically consequential, since chronic inflammatory adhesions and a distorted Calot triangle raise the risk of bile duct injury during cholecystectomy [1], [9].

The patient described here departs from the usual profile in several respects that carry practical implications. She was 33 years old, which places her well below the age at which Mirizzi syndrome is ordinarily anticipated. Her illness followed an acute-on-chronic course, in which several months of biliary pain gave way to acute cholangitis with severe obstructive jaundice. Her admission bilirubin and CA 19-9 were high enough to raise concern about a biliary malignancy, although no neoplastic mass was demonstrated. Operative exploration confirmed a cholecystocholedochal fistula consistent with Csendes type II to III disease, indicating that mural erosion had already occurred. Finally, the case illustrates the potential role of percutaneous transhepatic biliary drainage (PTBD) as a bridge to definitive surgery in a centre where hepatobiliary surgery and interventional radiology operate jointly. The report aims to raise awareness of advanced Mirizzi syndrome in young patients, to describe the diagnostic pitfalls that mimic biliary malignancy, and to correlate the radiological, operative and histopathological findings within a staged multidisciplinary pathway.

2 Case Report

A 33-year-old woman presented with right upper quadrant abdominal pain radiating to the back. The pain had been present for six months and had worsened markedly during the preceding week. She also described progressive jaundice, malaise, anorexia, nausea and recurrent vomiting, together with intermittent fever, acholic stools and tea-coloured urine, a combination indicating significant biliary obstruction. She had lost 4 kg over the previous month. Dietary history was notable for habitual consumption of fatty food. There was no history of previous biliary surgery, instrumentation or known liver disease.

On examination, she was alert and orientated, with blood pressure 120/80 mmHg, pulse rate 80 beats per minute, respiratory rate 20 breaths per minute and temperature 36.0 degrees Celsius. She appeared pale and lethargic and was overtly jaundiced. Abdominal examination elicited a positive Murphy sign, in keeping with cholecystitis. The relatively preserved haemodynamic parameters contrasted with the severity of the biochemical derangement, a discrepancy that can delay recognition of high-grade biliary obstruction (Figure 1).





Figure 1: Clinical appearance of a 33-year-old woman with progressive right upper quadrant pain and jaundice. (A) Conjunctival and scleral icterus. (B) Mild abdominal distension, consistent with severe biliary obstruction and hepatobiliary inflammation.

Laboratory investigation confirmed severe conjugated hyperbilirubinaemia with total bilirubin 16.4 mg/dL and direct bilirubin 13.5 mg/dL. Aspartate aminotransferase was 171 U/L and alanine aminotransferase 106 U/L. The leukocyte count was 12,700/mm3 and C-reactive protein was 2.86 mg/dL, indicating an acute inflammatory process superimposed on chronic biliary disease. Haemoglobin was 8.3 g/dL, sodium 132 mmol/L and potassium 3.1 mmol/L, and serum albumin was reduced, a profile consistent with chronic undernutrition and ongoing catabolic stress. Carcinoembryonic antigen was within normal limits whereas CA 19-9 was raised to 234 U/mL. Applying the Tokyo Guidelines 2018 criteria, the combination of systemic inflammation, cholestasis and biliary dilatation with a demonstrable cause on imaging established a definite diagnosis of acute cholangitis, and the leukocyte count above 12,000/mm3 together with total bilirubin above 5 mg/dL placed the episode in the moderate (grade II) severity category, for which early biliary drainage is indicated [15,16]. The serial laboratory course is summarised in Table 2.

Abdominal ultrasonography demonstrated a well-defined heteroechoic lesion with regular margins measuring approximately 1.73 cm at the level of the common hepatic duct, accompanied by dilatation of the right and left intrahepatic bile ducts. No convincing distal obstructing lesion was identified (Figure 2).

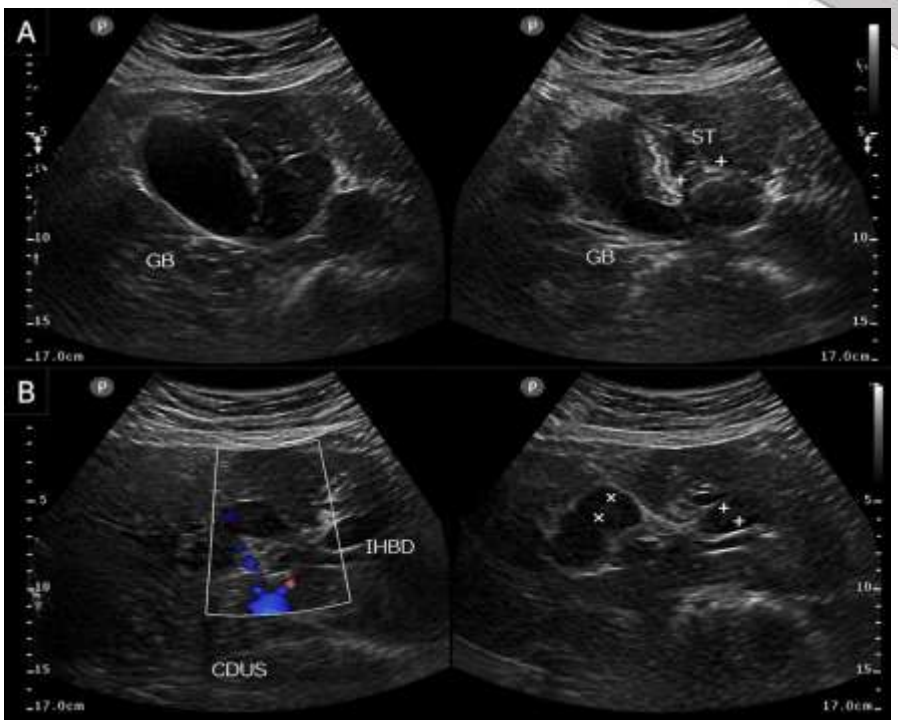


Figure 2: Abdominal ultrasonography. (A) Gallbladder (GB) containing an echogenic focus consistent with a stone (ST), measured with electronic calipers at approximately 1.73 cm. (B) Colour Doppler ultrasonography (CDUS) showing the dilated intrahepatic bile ducts (IHBD). No distal obstructing lesion was demonstrated

Contrast-enhanced computed tomography showed an enlarged liver containing multiple non-enhancing fluid attenuation lesions measuring 0.3 to 1.5 cm, thickening of the common bile duct wall, and a thick-walled gallbladder containing sludge. The pancreas appeared normal and no soft tissue mass was identified at the hepatic hilum or within the gallbladder fossa. The study was interpreted as cholangitis with hepatic microabscesses, hepatomegaly and cholecystitis with sludge; the tip of the percutaneous biliary drain is visible within the common hepatic duct on this examination (Figure 3).

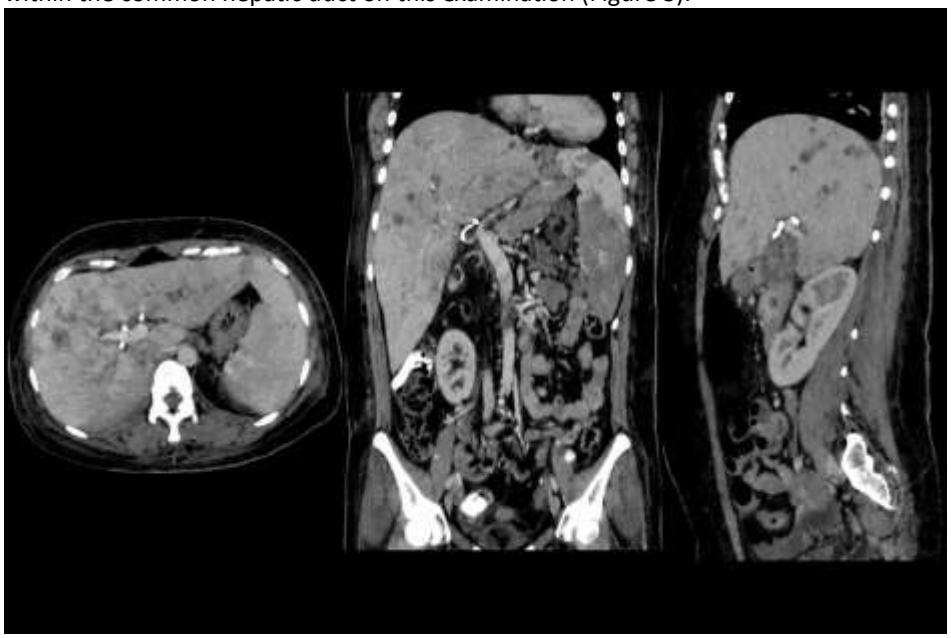


Figure 3: Abdominal computed tomography showing an enlarged liver with multiple non-enhancing fluid attenuation lesions measuring 0.3 to 1.5 cm, thickening of the common bile duct wall, and a thick-walled gallbladder containing sludge, with the pigtail tip of the percutaneous biliary drain in the common hepatic

duct. The pancreas is normal. Main findings: cholangitis with hepatic microabscesses, hepatomegaly, cholecystitis with sludge, and a biliary drain in situ

Given the severity of the cholangitis and the degree of jaundice, biliary decompression was undertaken by the interventional radiology service. Under real-time ultrasound guidance, a right intrahepatic duct was punctured and an 8.5 French catheter was advanced into the biliary tree. Fluoroscopic cholangiography opacified the right intrahepatic ducts and the proximal common hepatic duct and demonstrated extrinsic compression of the common hepatic duct by a stone impacted at the gallbladder neck, with dilatation of the right and left intrahepatic ducts. A filling defect was identified at the hilar segment, attributed to intraductal clot rather than to a further calculus, and was cleared by aspiration through the catheter. Thick bile drained freely thereafter (Figure 4). An enlarged left ovary with features of pelvic venous congestion was noted incidentally on the pelvic images and was managed conservatively.

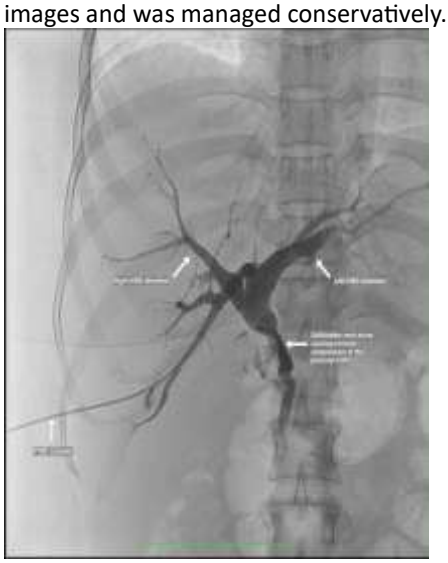


Figure 4: Percutaneous transhepatic biliary drainage. Contrast opacifies the right intrahepatic bile ducts and the proximal common hepatic duct. A stone impacted at the gallbladder neck compresses the proximal common hepatic duct extrinsically, producing dilatation of the right and left intrahepatic bile ducts. The annotations on the image are in Indonesian; their English equivalents are as follows. Dilatasi IHBD kanan, dilated right intrahepatic bile duct. IHBD kanan, right intrahepatic bile duct. Dilatasi IHBD kiri, dilated left intrahepatic bile duct. IHBD kiri, left intrahepatic bile duct. CHD proksimal, proximal common hepatic duct. Batu di neck GB yang mendesak CHD proksimal dari luar, stone at the gallbladder neck compressing the proximal common hepatic duct from outside the duct lumen. Kateter PTBD, percutaneous transhepatic biliary drainage catheter.

Biliary decompression produced a rapid biochemical response. Total bilirubin fell from 16.4 mg/dL to 6.2 mg/dL within several days, the leukocyte count and C-reactive protein declined, and CA 19-9 decreased in parallel with the resolution of cholestasis. Nausea settled, appetite improved and the patient became suitable for definitive surgery. No haemorrhagic, septic or catheter-related complication occurred during the drainage period.

Open cholecystectomy with common bile duct exploration was subsequently performed. The gallbladder was contracted with a markedly thickened wall and was embedded in dense inflammatory adhesions requiring careful adhesiolysis. The common bile duct was dilated and a communication between the gallbladder and the common bile duct was identified, confirming a cholecystocholedochal fistula corresponding to Csendes type II to III. A distal ductal stone was extracted with stone forceps and choledochoscopy confirmed patency of the duct both proximally and distally. The fistulous defect was repaired using a gallbladder remnant flap and a T-tube was placed for controlled biliary drainage (Figure 5).



Figure 5: Open cholecystectomy with common bile duct exploration. Left: operative field showing the dilated common bile duct opened after adhesiolysis, with the gallbladder communicating with the duct through a fistula. Upper right: the extracted calculus. Lower right: the resected gallbladder specimen with a thickened wall. Choledochoscopy confirmed proximal and distal ductal patency after clearance, and a T-tube was placed.

Histopathological examination of the gallbladder showed chronic cholecystitis with acute exacerbation, characterised by prominent Rokitansky-Aschoff sinuses and dense inflammatory cell infiltration. No dysplasia or malignancy was identified. The coexistence of long-standing mural inflammation with an established biliary fistula documents the progression of the disease from uncomplicated cholelithiasis to advanced Mirizzi syndrome (Figure 6).

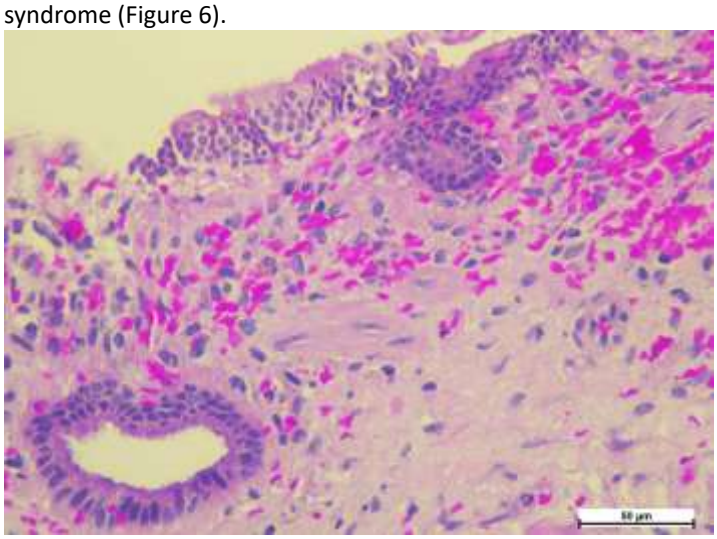


Figure 6: Histopathology of the gallbladder showing chronic cholecystitis with prominent Rokitansky-Aschoff sinuses and inflammatory cell infiltration. No malignancy was identified.

Perioperative supportive care comprised red cell transfusion, albumin replacement, intravenous fluid and electrolyte correction, and broad-spectrum antibiotic therapy. Recovery was uneventful. Pain and nausea resolved, mobilisation progressed satisfactorily and biochemical parameters continued to improve, with total bilirubin declining to 3.3 mg/dL, near-normalisation of the transaminases and a rise in haemoglobin. The percutaneous drain remained patent throughout and no major postoperative complication occurred. The patient was discharged in good clinical condition.

3 Discussion

3.1 Pathogenesis and the significance of early onset

Mirizzi syndrome begins as extrinsic compression of the common hepatic duct by a stone impacted at the gallbladder neck or in the cystic duct. Persistent mechanical pressure generates a localised inflammatory response with mucosal ulceration and fibrosis, and continued erosion may ultimately create a communication between the gallbladder and the bile duct [2], [10]. The Csendes classification grades this sequence according to the proportion of the ductal circumference involved: type I denotes extrinsic compression without fistula, type II a fistula involving less than one third of the circumference, type III a fistula involving up to two thirds, and type IV complete destruction of the ductal wall [11]. Types Va and Vb were later added to describe cholecystoenteric fistula without and with gallstone ileus [9]. The operative findings in the present patient, in which a fistulous communication was present but the duct remained reconstructible over a T-tube using a gallbladder flap, correspond to the type II to III range.

The interval required for this sequence to develop explains why the syndrome is usually a disease of later life. Reported series describe a mean symptom duration of three to twenty-four months before diagnosis and a clear predominance of middle-aged and older women [1]. Documented progression to a mature fistula at 33 years of age is therefore unusual and implies either accelerated stone formation or a prolonged, poorly recognised symptomatic period. The dietary history of habitual high fat intake is consistent with an early onset of gallstone disease, and cholelithiasis is itself common in this region, with reported prevalence of 3% to 10% in Asian populations [12]. Anatomical predisposition also merits consideration, since a long cystic duct running parallel to the common hepatic duct with low insertion increases the surface of contact available for pressure necrosis [2]. No such anatomical variant was documented in this patient, and this mechanism therefore remains speculative in her case.

3.2 Clinical and biochemical profile

The presentation was that of chronic biliary disease with acute decompensation. Six months of right upper quadrant pain followed by a week of rapid deterioration, jaundice, vomiting and weight loss corresponds closely to the symptom profile reported in large series, in which abdominal pain occurs in 65.7% to 100% and jaundice in 45% to 87.5% of patients, and a positive Murphy sign is found in about half [1]. Anaemia at 8.3 g/dL with hypoalbuminaemia reflected the chronic phase of the illness, while the leukocytosis and raised C-reactive protein reflected the acute infective component.

The degree of hyperbilirubinaemia deserves particular concern. Mean total bilirubin values reported across Mirizzi series generally lie between 2 and 9.9 mg/dL [1], so an admission value of 16.4 mg/dL places this patient at the severe end of the spectrum and is consistent with advanced disease complicated by cholangitis. Applying the Tokyo Guidelines 2018 severity criteria, two of the five parameters were satisfied on admission, establishing grade II disease and providing the formal indication for early drainage [13], [14]. Hyponatraemia and hypokalaemia were attributed to reduced intake with vomiting and were corrected before operation.

3.3 CA 19-9 as a diagnostic pitfall

An elevated CA 19-9 in a jaundiced patient frequently prompts consideration of cholangiocarcinoma or gallbladder carcinoma, and this concern is not unreasonable, since Mirizzi syndrome and gallbladder cancer coexist in around 5% of operated patients [15]. The marker is nonetheless a poor discriminator in the presence of biliary obstruction. In a study of 164 patients with a raised CA 19-9, the median value in benign disease was 102 U/mL with an interquartile range of 50 to 264 U/mL, compared with 910 U/mL in pancreatobiliary malignancy, and the degree of elevation correlated with serum bilirubin among jaundiced patients with benign disease [4]. In a longitudinal series of 128 patients with obstructive jaundice, CA 19-9 exceeded 37 U/mL in 61% of benign cases and fell after biliary drainage in almost all of them, whereas persistence above 90 U/mL after successful decompression favoured a malignant cause [5].

The value of 234 U/mL recorded here falls within the benign interquartile range described by Mann and colleagues, and its decline after percutaneous drainage followed the pattern expected of a reactive elevation. A value obtained after biliary decompression is therefore far more informative than one measured at the point of maximal cholestasis, and this case supports that practice. The absence of a mass lesion on cross-sectional imaging and the subsequent histopathological confirmation of benign disease resolved the question definitively.

3.4 Multimodality imaging

Ultrasonography served its conventional role as the initial examination, demonstrating a calcific focus at the level of the common hepatic duct with bilateral intrahepatic duct dilatation, while leaving the precise mechanism of obstruction unresolved. This limitation is well described: reported sensitivity for Mirizzi syndrome ranges from about 10% to 50% across series, with 48% in the Mayo Clinic experience of 36 patients [1], [6]. Computed tomography added the information that mattered most at that stage by excluding a hilar or gallbladder mass and

by identifying hepatic microabscesses, which established the severity of the infective process. Several authors emphasise that the principal contribution of computed tomography in this setting lies in the exclusion of malignancy more than in direct visualisation of the compressed duct [1], [8].

Percutaneous cholangiography provided the definitive preoperative demonstration of the mechanism, showing a smooth extrinsic indentation of the common hepatic duct produced by a stone lying outside the duct lumen. Direct cholangiography, whether endoscopic or percutaneous, remains the most reliable means of demonstrating the level of obstruction and of identifying a cholecystobiliary communication [1], [16]. Magnetic resonance cholangiopancreatography was not obtained in this patient because the clinical situation required urgent decompression; where it is available and the patient is stable, it offers a non-invasive alternative with a reported sensitivity of approximately 80% and specificity of 83% [7], and combined magnetic resonance and computed tomography assessment increases the preoperative detection rate [1]. The contribution of each modality in this case is summarised in Table 1.

Table 1: Contribution of each imaging modality in the present case, with reported diagnostic performance in Mirizzi syndrome

Modality	Findings in this patient	Contribution to management	Reported performance
Abdominal ultrasonography	Heteroechoic focus of about 1.73 cm at the level of the common hepatic duct; dilated right and left intrahepatic ducts; no distal obstructing lesion	Established the presence and level of biliary obstruction and prompted cross-sectional imaging	Sensitivity approximately 10% to 50%; 48% in a 23-year single-centre series [1], [6]
Computed tomography	Hepatomegaly with multiple non-enhancing lesions of 0.3 to 1.5 cm; common bile duct wall thickening; thick-walled gallbladder with sludge; normal pancreas; no mass	Excluded a hilar or gallbladder mass and identified hepatic microabscesses, defining the severity of infection	Sensitivity comparable to ultrasonography; principal value lies in excluding malignancy [1], [8]
Percutaneous transhepatic cholangiography	Extrinsic compression of the common hepatic duct by a stone at the gallbladder neck; bilateral intrahepatic duct dilatation; hilar filling defect attributed to clot	Confirmed the mechanism and level of obstruction while simultaneously providing therapeutic decompression	Direct cholangiography is the most reliable preoperative demonstration of level and of cholecystobiliary communication [1], [16]
Magnetic resonance cholangio-pancreatography	Not performed; urgent decompression took priority	Where the patient is stable, provides non-invasive ductal mapping before intervention	Sensitivity approximately 80%, specificity 83% [7]; combined use with computed tomography raises detection [1]
Intraoperative assessment with choledochoscopy	Cholecystocholedochal fistula, dilated common bile duct, distal ductal stone, patent duct after clearance	Confirmed the diagnosis and determined the reconstructive technique	Remains the reference standard; 12% to 84% of cases are diagnosed only at operation across series [1], [17]

3.5 Percutaneous biliary drainage as a bridge to definitive surgery

The Tokyo Guidelines 2018 recommend biliary drainage for acute cholangitis of moderate or severe grade, with endoscopic transpapillary drainage as the first-line technique and percutaneous transhepatic drainage as the alternative when the endoscopic route is unavailable or unsuitable [14], [18]. In Mirizzi syndrome, endoscopic cannulation may be technically demanding because the obstruction is extrinsic and the hilar anatomy is distorted by chronic inflammation, and the percutaneous route offers direct access above the level of obstruction. In the present case, severe cholangitis with hepatic microabscesses and grade II severity made prompt decompression the priority, and PTBD provided it within a single session.

The physiological effect was immediate and measurable. Total bilirubin fell by more than 60% within several days, inflammatory markers declined and nutritional support could be delivered to a patient who had been vomiting and anorexic for a week. Operating on a decompressed, non-septic biliary tree is preferable to operating during active cholangitis in a hostile hilum, and the same principle of preoperative decompression followed by definitive repair has been described for endoscopic drainage in high-grade Mirizzi syndrome [19]. Interventional radiology also contributes diagnostically, and delayed recognition of Mirizzi syndrome in the interventional setting is a recognised pattern worth guarding against [20].

The technique itself carries defined risks that were anticipated and managed. Haemorrhagic adverse events, of which haemobilia is the most frequent manifestation, complicate a minority of procedures, and haemobilia severe enough to require transcatheter embolisation was reported in 2.3% of 346 drainages, with a lower rate for right-sided than for left-sided access [21]. Hepatic arterial injury is less common still, occurring in fewer than 1% of a series of 3,110 procedures [22]. The hilar filling defect observed at cholangiography in this patient was consistent with intraductal clot and resolved with catheter aspiration, without transfusion for biliary bleeding or angiographic intervention. Ultrasound guidance at the moment of puncture, together with correction of coagulation status beforehand, remains the principal safeguard, supported by periprocedural antibiotic cover.

3.6 Operative and histopathological correlation

At laparotomy the gallbladder was contracted and thick-walled and lay within dense adhesions, a configuration described by Beltran as characteristic of the distorted anatomy of Mirizzi syndrome and one that renders dissection of Calot triangle hazardous [10]. Open surgery remains the preferred approach when a cholecystobiliary fistula is present, although laparoscopic management by experienced biliary teams has been reported for type II disease [23], [24]. For fistulas involving less than two thirds of the ductal circumference, partial cholecystectomy with repair of the defect over a T-tube using the gallbladder remnant is the technique generally recommended, reserving bilioenteric anastomosis for complete ductal destruction [2], [17]. The procedure performed here followed that principle.

Histopathology closed the diagnostic loop. Chronic cholecystitis with acute exacerbation, prominent Rokitansky-Aschoff sinuses and dense inflammatory infiltration provided tissue evidence of the long-standing inflammatory process inferred from the imaging, and the absence of malignancy confirmed that the raised CA 19-9 had been reactive. Correlation of this kind is particularly valuable when a preoperative tumour marker has raised the possibility of carcinoma, because published series document coexistent gallbladder cancer in a small but clinically important proportion of patients with Mirizzi syndrome [15].

3.7 Incidental pelvic findings

An enlarged left ovary with features of pelvic venous congestion was identified on the same examination. No causal or pathophysiological relationship with Mirizzi syndrome has been established, and the finding is reported here as an incidental observation rather than as an associated feature. It is mentioned because incidental pelvic findings in a young woman investigated for jaundice may require separate follow-up, and because attributing significance to coincidental findings in single-patient reports risks introducing spurious associations into the literature.

3.8 A stepwise approach derived from this case

The management sequence used here can be summarised as a practical pathway for comparable presentations, recognising that recommendations derived from a single patient carry limited evidential weight.

A. Maintain suspicion. Consider Mirizzi syndrome in any patient with obstructive jaundice and right upper quadrant pain, irrespective of age. A published scoring system combining symptom duration, jaundice, bilirubin above 1.2 mg/dL, leukocytosis, alkaline phosphatase above 150 U/L and radiological features such as intrahepatic biliary dilatation and the meniscus sign achieved 90.6% sensitivity at a threshold of three points and may assist preoperative identification [25].
B. Sequence the imaging. Begin with ultrasonography, proceed to computed tomography to exclude a mass and to assess hepatic complications, and obtain magnetic resonance cholangiopancreatography when the patient is stable enough to allow non-invasive ductal mapping before intervention [1], [7].
C. Grade the cholangitis and drain accordingly. Apply the Tokyo Guidelines 2018 criteria at admission. For grade II or III disease, arrange drainage early; where the endoscopic route is unfavourable or the hilar anatomy is distorted, percutaneous transhepatic drainage may be considered as an alternative route to access the biliary tree above the obstruction [14], [18].
D. Optimise before operating. Use the interval after drainage to correct anaemia, hypoalbuminaemia and electrolyte disturbance, to complete antibiotic therapy, and to document a falling bilirubin. Reassess CA

19-9 only after decompression, since values obtained during maximal cholestasis are difficult to interpret [5].

E. Plan the operation for the anatomy encountered. Anticipate dense adhesions and a distorted Calot triangle. Where a fistula involves less than two thirds of the ductal circumference, partial cholecystectomy with repair over a T-tube is appropriate; complete ductal destruction calls for bilioenteric reconstruction [2], [17].
F. Follow up deliberately. Confirm drain function and the absence of bile leak after operation, document normalisation of liver biochemistry and CA 19-9, and image the biliary tree before removing the T-tube to exclude retained stones or a developing stricture.

3.9 Limitations

This report describes a single patient and its conclusions are correspondingly provisional. Magnetic resonance cholangiopancreatography was not performed, so the comparison between non-invasive and direct cholangiography rests on published data rather than on paired imaging in this patient. Several laboratory values were not recorded at every time point, which limits the completeness of the biochemical trend presented in Table 2. Follow-up was confined to the index admission, so late outcomes such as anastomotic stricture, recurrent stone formation and long-term biliary function are not reported. Finally, the classification of the fistula as Csendes type II to III was made on operative inspection without formal measurement of the proportion of the ductal circumference involved, a limitation inherent in the intraoperative application of this classification [1].

Table 2: Serial laboratory values at admission, after percutaneous transhepatic biliary drainage and after definitive surgery.

Parameter	Reference range	Admission	After PTBD	After surgery
Total bilirubin (mg/dL)	0.3–1.0	16.4	6.2	3.3
Direct bilirubin (mg/dL)	<0.3	13.5	Not recorded	2.4
Aspartate aminotransferase (U/L)	<40	171	Not recorded	Near normal
Alanine aminotransferase (U/L)	<41	106	Not recorded	Near normal
Leukocytes (per mm3)	4,000–11,000	12,700	Decreased	Normal
C-reactive protein (mg/dL)	<0.5	2.86	Decreased	Not recorded
Haemoglobin (g/dL)	12.0–15.0	8.3	Not recorded	Increased
Sodium (mmol/L)	135–145	132	Not recorded	Corrected
Potassium (mmol/L)	3.5–5.1	3.1	Not recorded	Corrected
CA 19-9 (U/mL)	<37	234	Decreased	Normal
Carcinoembryonic antigen	Institutional range	Normal	Not recorded	Not recorded

Reference ranges are indicative and should be replaced with the values used by the reporting laboratory. Entries recorded as decreased, increased, corrected or near normal reflect the descriptive terms available in the clinical record; numerical values should be inserted where they can be retrieved. PTBD, percutaneous transhepatic biliary drainage.

4 Conclusion

This case documents an uncommon presentation of Mirizzi syndrome in a 33-year-old woman whose disease had progressed to a cholecystocholedochal fistula consistent with Csendes type II or III. Age alone should not exclude the diagnosis when progressive obstructive jaundice, right upper quadrant pain, and systemic inflammatory findings occur together.

Diagnostically, the case illustrates the complementary contributions of ultrasonography, computed tomography, and direct cholangiography. Ultrasonography and computed tomography identified biliary abnormalities and demonstrated no identifiable mass lesion, while fluoroscopic cholangiography provided direct evidence of extrinsic compression of the common hepatic duct. The elevated CA 19-9 observed during marked cholestasis, together with the absence of malignancy in the examined gallbladder specimen, supported a benign explanation for the tumour marker elevation, although its interpretation requires caution in the setting of biliary obstruction.

Percutaneous transhepatic biliary drainage served as both a diagnostic adjunct and a bridge to definitive surgery, providing effective biliary decompression and clinical stabilisation before surgical intervention. Subsequent open cholecystectomy with common bile duct exploration confirmed the cholecystobiliary fistula and relieved the

biliary obstruction during the reported hospitalisation. Histopathological examination demonstrated chronic inflammatory changes without evidence of malignancy in the examined specimen.

Taken together, this case highlights the importance of early clinical suspicion, multimodality imaging, and multidisciplinary coordination in the management of advanced Mirizzi syndrome. It also illustrates the potential role of PTBD as a bridge to definitive surgery in selected patients with acute cholangitis when percutaneous biliary drainage is considered appropriate by the multidisciplinary team. Further evidence is needed before broader conclusions can be drawn regarding the comparative effectiveness of this staged approach relative to endoscopic drainage.

4.1 Clinical implications

This case suggests that an integrated interventional radiology and surgical pathway may be a reasonable option in selected patients with complex biliary obstruction, particularly where advanced endoscopic expertise is not continuously available. Percutaneous drainage may be considered in severe cholangitis or in Mirizzi syndrome with distorted anatomy, although its effect on operative safety and morbidity cannot be established from a single case.

4.2 Future directions

Further work is needed to define imaging criteria for the early detection of Mirizzi syndrome in younger patients and to establish where minimally invasive biliary decompression should sit within the treatment algorithm that precedes definitive surgery. Multicentre collection of cases would be required, given the rarity of the condition and the heterogeneity of current reporting.

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